Clinical review

Fortnightly review

Corticosteroid injections in tendon lesions

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Periarticular soft tissue rheumatic complaints include localised disorders of tendons, ligaments, muscles, fascia, and joint capsules. Such complaints account for up to 59% of new patient referrals to a rheumatology practice[1] and up to 15% of consultations in primary care.[2] Tendon lesions (tendinopathies) represent a large proportion of these complaints and are often the most difficult to treat. Their high incidence of chronicity and recurrence[3 4] result in appreciable morbidity and loss of productivity, representing a major socioeconomic burden.[5]

Corticosteroid injections are one of the most commonly used treatments for chronic tendon lesions.[1] Despite their popularity, the rationale for use of corticosteroid injections is contentious, the evidence for benefit is lacking, and they have potential adverse effects. This article describes the nature of tendon lesions, the basis for the use of local corticosteroids, and current evidence for beneficial and adverse effects.

Summary points

- Tendon lesions are a varied group of common complaints
- Various treatments are available
- There is no good evidence to support the use of local corticosteroid injections in chronic tendon lesions
- The lack of evidence is due to a true lack of effect or a lack of good trials
- Accuracy of diagnosis and injection are important.

Methods

I searched the Cochrane Library, Medline, and Embase to identify studies of common pathological findings in tendinopathies and treatment with corticosteroids. I also looked for good systematic reviews of randomised controlled trials, and good randomised controlled trials published since the search date of identified reviews, relating to the evidence for effect, guidelines for use, and potential adverse effects of corticosteroid injections in tendinopathies. When no systematic review was available, I used data from individual randomised controlled trials, applying Sackett et al's quality criteria.[6]

Tendon structure and pathology

Tendons are collagenous structures with additional tenocytes, water, and other matrix components. Tendons are surrounded by loose connective tissue, the paratenon, which forms an elastic sleeve that allows free movement of the tendon. Where tendons travel through narrow areas (around the hands and feet), this tissue becomes specialised into a tenosynovial sheath, helping to reduce friction between the tendon and surrounding structures.[7]

The term tendinopathy encompasses a spectrum of disorders, including lesions of the tenosynovium the paratenon, the enthesis, or tendon proper (table 1). Lesions can coexist, and the tendon can also tear partially or completely (fig 1).

Pain and dysfunction are the main symptoms. Clinical signs such as swelling or thickening of the tendon are variable. Local tenderness and exacerbation of pain with testing against resistance and with passive stretching are common to most lesions. Appreciable weakness suggests a torn or ruptured tendon. Crepitus occurs in severe paratenonitis, while triggering and a palpable tendon nodule suggest tendinosis.

Differentiating between paratenonitis and tendinosis is often difficult, and classic signs of inflammation are often absent. Tendinopathies are also described according to the duration of symptoms; a commonly used empirical classification is acute (up to 2 weeks in duration), subacute (2-4 weeks), and chronic (over 6 weeks).[8]

With the exception of some cases of tenosynovitis, most histological studies of chronic tendon lesions have shown degenerative features with no cellular evidence for inflammation.[7] The term tendinosis (degeneration) is therefore preferred to that of tendinitis, which suggests inflammation. Such degenerative features may be asymptomatic.[9]

Published histopathological studies all have limitations. The presence or absence of biochemical features of inflammation has been largely ignored (although normal prostaglandin concentrations have been

Table 1 Common types of tendon lesion

Disorder	Description	Example	Clinical signs*
Paratenonitis	Disorder of paratenon layer covering tendon	Achilles' paratenonitis	Pain, tenderness, diffuse swelling, crepitus, warmth (early)
Tenosynovitis†	Disorder of paratenon sheath and its synovial lining	De Quervain's tenosynovitis	Pain, tenderness, swelling in sheath, crepitus, warmth (early)
Tendinosis	Intratendinous degeneration	Lateral epicondylitis, rotator cuff tendinosis	Pain, point tenderness, palpable nodule
Enthesopathy	Disorder of tendon as it inserts on to bone	Insertional Achilles' tendinosis	Tenderness, swelling at tendon insertion
Tear	Disruption of the integrity of tendon. Can be partial or full thickness	Rotator cuff tear	Pain (may be absent), weakness, with or without palpable gap

*Clinical signs are variable and overlapping. Most patients exhibit tenderness and pain on resisted testing of movement and on stretching.
†A form of paratenonitis.

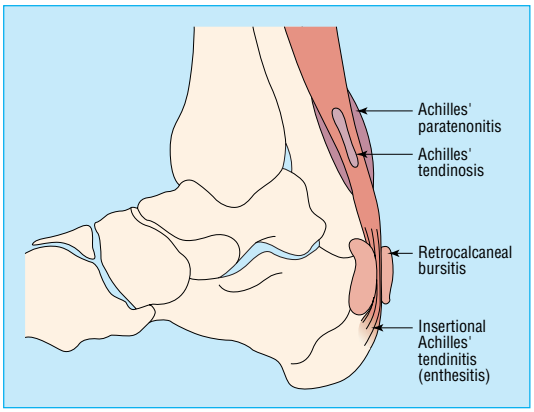

Fig 1 Different types of lesions—for example, around Achilles' tendon—can be difficult to distinguish

shown in painful lesions of the Achilles' tendon[10]) and most studies have been limited to end stage, non-healing lesions.[11–13] It is not clear whether the degenerative features are preceded by an inflammatory phase. The pathology may also vary according to the site of the lesion.[9 14]

Other mechanisms of tendon pain

In the absence of inflammation, other factors may cause a painful tendon. These include irritation of mechanoreceptors by vibration, traction, or shear forces[9 12–15] and triggering of nociceptive receptors by neurotransmitters such as substance P and by biochemical irritants such as chondroitin sulphate.[9 16 17]

Tendinopathies are an extremely varied group of lesions, and clinical differentiation between lesions can be difficult. Although the underlying pathology in chronic lesions is degenerative, inflammation may have a role in earlier lesions.

Effects of local corticosteroid injections

Local corticosteroids are used to reduce inflammation in patients with chronic tendinopathies.[18] However, inflammation is not a major feature in many of these lesions and, if present, is a vital component of the healing response. Inhibiting this process may result in a suboptimal outcome.[19] Other mechanisms of pain reduction by corticosteroids in tendon lesions have not been established.

Studies on animal models have shown that intratendinous corticosteroid adversely affects the biomechanical properties of tendons.[20 21] Corticosteroids can inhibit formation of adhesions, granulation, and connective tissue; reduce tendon mass; and decrease biomechanical integrity and the amount of load that can be taken before failure.[20 21] The biomechanical effects of peritendinous corticosteroid on human tendons are unestablished. However, case reports of rupture of tendons after injection are common.[22 23]

Clinical evidence

Trigger finger, tennis elbow, Achilles' tendinopathy, and rotator cuff lesions are three of the most common tendinopathies, and many studies have evaluated corticosteroid injections in these three conditions.

Trigger finger

Trigger finger (fig 2) is one of the few conditions where evidence supports the use of corticosteroids.[24 25] In a double blind, randomised, placebo controlled trial, Murphy et al compared local injection of betamethasone plus lignocaine in 14 subjects with lignocaine alone in 10 control subjects.[24] At 3 weeks' follow up, 10 subjects in the treatment group were asymptomatic compared with two in the control group. In another double blind, placebo controlled trial of local injection of methylprednisolone acetate plus lignocaine with lignocaine alone in 41 subjects with trigger finger, Lambert et al found a significant improvement (physician rated) in 60% of the treatment group compared with 16% of the control group at 1 month follow up.[25]

Rotator cuff tendinopathy

Subacromial corticosteroid injections improve short term range of motion but not pain in patients with rotator cuff tendinosis.[26 27] Green et al identified two randomised placebo controlled trials of corticosteroid

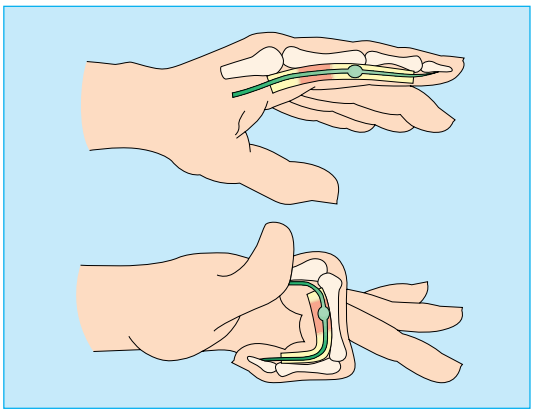

Fig 2 Trigger finger: entrapment fo the flexor tendon due to thickening and tendinosis

injections for rotator cuff tendinosis with a four week follow up.[28 29] The pooled data (n=90) showed improvement in abduction at four weeks (weighted mean difference 35°, 95% confidence interval 14° to 55°) but there was no effect on pain (7%, −33% to 47%).[26 27] A randomised controlled trial of subacromial methylprednisolone and lignocaine found it was no better than lignocaine alone at 12 weeks in 55 subjects with rotator cuff tendinosis[27 30] Effectiveness in the longer term is unknown, and there is insufficient good data on which to base a recommendation for practice.

Tennis elbow

Assendelft et al performed a systematic review of validity and outcome of randomised controlled trials of corticosteroid injections for lateral epicondylitis.[31] They identified 12 trials with an adequate methodological score, although overall the quality was poor to moderate, with a median methodological score of 40/100. Effectiveness of treatment in these studies was assessed by the change in pain score or a global assessment by the patient or an assessor. Pooled analysis indicated short term effectiveness only (2-6 weeks), with a pooled odds ratio of 0.15 (95% confidence interval 0.1 to 0.23), $\chi^2=13.3$ (df=5), indicating statistical heterogeneity. At longer term follow up no difference in outcome was found between treatment and control groups. Studies with a higher methodological score had more favourable results. No conclusions could be made about the most suitable corticosteroid, dose, injection interval, or injection volume.

Hay et al did a multicentre pragmatic randomised controlled trial in 164 subjects presenting with a new episode of lateral epicondylitis, comparing local corticosteroid injection, oral non-steroidal anti-inflammatory drugs, and simple analgesics.[32] About a third of patients in each group had symptoms for more than three months. After four weeks, 82% of patients were "better" (pain ⩽ 3 on patient's 10 point Likert scale) in the corticosteroid group compared with 48% in the non-steroidal anti-inflammatory group and 50% in the analgesic groups. However, at 1 year, outcome was similar in all groups (84% *v* 85% *v* 82%).

Achilles' tendinopathy

The only double blind randomised controlled trial examining the effectiveness of corticosteroid injections in Achilles' paratenonitis showed no benefit (pain, tenderness, and return to normal activity) of peritendinous methylprednisolone (40 mg) and marcaine over marcaine injections alone in 28 subjects.[33]

Table 2 Comparison of injectable corticosteroids

Preparation	Potency*	Concentration (mg/ml)	Solubility (% wt/vol)	Dose (mg)
Short acting:				
Hydrocortisone acetate	1	25	0.002	5-25
Moderate acting:				
Prednisolone acetate	4	25	NA	5-25
Methylprednisolone acetate	5	20, 40, 80	0.001	5-40
Long acting				
Tramcinalone acetonide	5	10, 40	0.004	5-40
Triamcinalone hexacetonide†	5	20	0.0002	NA

NA=not available.
*Hydrocortisone equivalents (per mg).
†Insolubility makes it unsuitable for use in soft tissue injections.

Interpreting the literature

There is no good evidence to support the use of local corticosteroid injections in chronic tendon lesions. This is due to either a true lack of effect or a lack of good trials. Trigger finger is notably one of the few tendon lesions where there is evidence of chronic inflammation[9]; this may explain the improvement found with local corticosteroids. Methodological issues that may have affected clinical research into the efficacy of corticosteroid injections include study design and accuracy of both diagnosis and injection.

Eustace et al showed that, even in the hands of musculoskeletal specialists, only a minority of injections for shoulder pain are performed accurately (29% of subacromial and 42% of intra-articular injections) and that outcome significantly correlates with accuracy of injection.[34] Zhingis et al found similar results in subjects with tenosynovitis stenosans (De Quervain's).[35] For this reason, ultrasound guided injection is becoming popular.[36]

Many of the studies have had inadequate design. Problems include small sample sizes, unsuitable outcome measures, short term follow up, inadequate blinding, lack of a true placebo, and the inclusion of heterogeneous study populations.[26 27 31] The increasing availability of magnetic resonance imaging and ultrasonography should improve diagnostic reliability.

Potential adverse effects

The overall incidence of side effects after local corticosteroid injection for tendon lesions is unknown. Similarly, the relevance of the steroid used, the tissue affected, the extent of the injury, the phase of healing at the time of injection, and postinjection events (particularly loading of the tissue) remains undetermined. Complete tendon rupture with loading after steroid injection is recognised,[22 23] although rigorous studies have not been performed.

Sepsis is reported in up to 1 in 17 intra-articular or soft tissue injections.[37] Other commonly reported side effects include tissue atrophy, facial flushing, postinjection flare, and hypersensitivity reactions.[18] Resuscitation facilities should be available in case patients have a rare severe reaction.[18]

Use of local corticosteroid injections in tendinopathies

Early intervention in tendon injuries aims to educate the patient, resolve the injury, and prevent chronicity and recurrence.[8 38] Since inappropriate immobilisation has deleterious consequences, including tissue atrophy,[39] the priority of medical management is to control pain to allow early rehabilitation. Initial management includes relative rest, pain control, support, stretching, appropriate exercise, and correction of provoking factors.[8 38] Failure to correct provoking factor often results in chronicity.[8 38]

Common approaches to controlling pain include simple analgesics, non-steroidal anti-inflammatories, acupuncture or dry needling, ice, ultrasound, and transcutaneous electrical nerve stimulation. Evidence to support these approaches is lacking.[26 27] Resting

Suggestions for use of local steroid injections in tendinopathies

- Reserve for chronic injuries, after intensive use of other approaches for at least 2 months has failed
- Use when rehabilitation is inhibited by symptoms
- Informed consent should be obtained from the patient, who must be willing to follow postinjection guidelines
- The practitioner should have full knowledge of the local anatomy
- Select the finest needle that will reach the lesion
- The practitioner's hands and the patient's skin should be cleansed and a no touch technique used
- Use short or medium acting corticosteroid preparations in most cases, with local anaesthetic
- Injection should be peritendinous; avoid injection into tendon substance
- Minimum interval between injections should be 6 weeks
- Use a maximum of three injections at one site
- Soluble preparations may be useful in those patients who have had hypersensitivity/local reaction to previous injection
- Details of the injection should be carefully recorded
- Do not repeat if two injections do not provide at least 4 weeks' relief

Postinjection advice

Warn the patient of early postinjection local anaesthesia and to avoid initial overuse
Advise resting for at least 2 weeks after injection and avoid heavy loading for 6 weeks
The patient should inform the doctor if there is any suggestion of infection or other serious adverse event

Contraindications to corticosteroid injection in soft tissue lesions

If pain relief and anti-inflammatory effects can be achieved by other methods
Local or systemic infection
Coagulopathy
Tendon tear
Young patients

splints, used intermittently, are often useful to prevent soft tissue contractures and prevent excessive movement during healing. Local anaesthetic injections may help diagnosis.[40]

Many of the recommendations for the use of local corticosteroid injections are based on anecdote. As is the case with intra-articular injections, there is no consensus with respect to environment (operating theatre, treatment room, clinic), the wearing of gloves, shaving of skin, the use of local anaesthetic, the safe number of injections at one site, or the appropriate interval between injections.[37] It is not surprising that evidence based guidelines do not exist. The box gives some suggestions on the use of corticosteroid injections for chronic tendinopathies.

Drugs have different potency and solubility, and solubility is inversely correlated with the duration of action (table 2).[18] There are few data on the absorption of corticosteroids from peritendinous injections, but methylprednisolone acetate remains in plasma for a mean of 16 days after periarticular injection.[41] Short or moderate acting, more soluble preparations (such as hydrocortisone and methylprednisolone) are recommended for soft tissue injections because in theory they cause fewer side effects; drugs with low solubility should not be used for soft tissue injections.[18]

Local anaesthetic is usually mixed with the corticosteroid in soft tissue injections to make the procedure more comfortable for patients and increase the volume of the injection for wider dispersion.[18 37 40] However, some manufacturers advise against mixing drugs because of the theoretical risk of clumping and precipitation of steroid crystals. There is no evidence that injection of local anaesthetic before the corticosteroid is beneficial, nor of a difference in outcome between long acting and short acting anaesthetics.[40]

Local steroid injections in the vicinity of the Achilles' or patellar tendon and in patients with tears are often discouraged because of concerns about rupture of heavily loaded tendons or impairment of tissue repair where disruption is already present[17 38] Again, evidence for these recommendations is lacking.

Summary

Tendinopathies are an important group of lesions with a broad spectrum of overlapping characteristics that can pose a diagnostic and therapeutic challenge. Although local corticosteroid injections are one of the most common treatments, there is no good evidence to support their use. This is due to either a true lack of effect or a lack of good trials. More research is warranted into the characteristics of specific tendinopathies and the biochemical, cellular, and clinical effects of corticosteroids on these lesions.

Competing interests: None declared.

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Lesson of the week

Causes of haematuria in adult polycystic kidney disease

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Do not assume that haematuria in association with adult polycystic kidney disease is always benign

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Patients with a chronic disease may develop new, but often related, symptoms. The assumption, however, that all symptoms are related and need no further investigation, may result in the misdiagnosis of a serious disease. Autosomal dominant polycystic kidney disease is a common inherited condition, occurring in between 1 in 200 and 1 in 1000 of the population.[1] After diagnosis patients are followed up by nephrologists because of the risk of progression to end stage renal failure. Renal function may be preserved, however, and outpatient attendance is often infrequent. If complications develop, affected individuals may present to primary care providers or a wide range of hospital and surgical specialties, where staff should be aware of possible diagnostic difficulties. Macroscopic haematuria is a common complication that is usually related to the polycystic kidney disease.[2] We present two cases where subsequent investigations discovered bladder carcinoma and suggest possible screening methods.

Case reports

Case 1—A 49 year old man with adult polycystic kidney disease and persistent microscopic haematuria since diagnosis nine years earlier, presented with a three week history of asymptomatic macroscopic haematuria. He was otherwise well with no reported anorexia or weight loss. Multiple urine cultures were sterile. Ultrasound scanning of the renal tract showed a three centimetre mass at the base of the bladder. At cystoscopy a papillary pT1 G2 tumour at the bladder neck was resected. There was no histological evidence of carcinoma in situ. The patient is currently undergoing treatment with intravesical BCG and remains well.

Case 2—A 34 year old woman with adult polycystic kidney disease was reviewed in the outpatient department shortly after she became pregnant. She was asymptomatic with no macroscopic haematuria, but a substantial number of red blood cells were discovered on microscopic urine examination. She had had several intermittent episodes of painless macroscopic haematuria for five years. These had settled with no intervention, and at her regular outpatient attendance the presence of haematuria had not been detected on urine dipstick or microscopy. Renal tract examination during fetal ultrasound scanning at eight weeks' gestation showed a non-mobile, lobulated soft tissue mass in the bladder. At cystoscopy a pTa G2 carcinoma was completely resected. No further specific treatment was indicated. The pregnancy continued to term, and the baby was delivered without complication.

Discussion

Almost two thirds (64%) of people with adult polycystic kidney disease also develop microscopic or macroscopic haematuria.[2] Most episodes are due to urinary tract infections and renal cyst rupture that relate to the underlying anatomical abnormalities. The symptoms are usually short lived[3] and resolve with conservative measures such as rest and antibiotic treatment. Renal stone disease is also common, occurring in 20% of patients.[4] However, macroscopic haematuria is also a presenting symptom of common, but unrelated, disor-